

## Anti-BID Antibody (6V154)

### Product Details

Ig Type:	Rabbit IgG
Reactivity:	Human
Conjugation:	Unconjugated
Clone:	6V154
Purification:	Protein A

### Applications

1. BID was immunoprecipitated using:
  - Lane A:0.5 mg Jurkat Whole Cell Lysate.
  - Lane B:0.5 mg RAW264.7 Whole Cell Lysate.
  - 2  $\mu$ L anti-BID rabbit polyclonal antibody and 15  $\mu$ L of 50 % Protein G agarose.
  - Primary antibody:
    - Anti-BID rabbit polyclonal antibody, at 1:200 dilution.
  - Secondary antibody:
    - Dylight 800-labeled antibody to rabbit IgG (H+L), at 1:5000 dilution.
  - Developed using the odyssey technique.
  - Performed under reducing conditions.
2. Anti-BID rabbit monoclonal antibody at 1:500 dilution.
  - Lane A: Jurkat Whole Cell lysate.
  - Lysates/proteins at 30  $\mu$ g per lane.
  - Secondary
    - Goat Anti-Rabbit IgG H&L (Dylight800) at 1/10000 dilution.
  - Developed using the Odyssey technique.
  - Performed under reducing conditions.
  - Predicted band size:22 kDa.
  - Observed band size:22 kDa

#### Verified Activity:

- Predicted band size: 22 kDa.
- Observed band size: 22 kDa.

Application: ELISA,IP,WB

Recommended WB: 1:500-1:2000; ELISA: 1:5000-1:10000; IP: 0.5-2  $\mu$ L/mg of lysate

### Properties

Stability & Storage:	Store at 2°C-8°C for 1 month. Store at -20°C or -80°C for 12 months. Avoid repeated freeze-thaw cycles. Preservative-Free.
Shipping:	Shipping with blue ice.

### Antigen Details

Immunogen: Recombinant Protein: Human BID protein (TMPY-02027)  
Antigen Species: Human  
Synonyms: BH3 interacting domain death agonist;AU022477;AI875481;2700049M22Rik

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### Research Background

The BH3 interacting domain death agonist (BID) is a pro-apoptotic member of the Bcl-2 protein family, which contains only the BH3 domain, and is required for its interaction with the Bcl-2 family proteins and for its pro-death activity. BID is important to cell death mediated by these proteases and thus is the sentinel to protease-mediated death signals. Recent studies further indicate that Bid may be more than just a killer molecule, it could be also involved in the maintenance of genomic stability by engaging at mitosis checkpoint. BID is an integrating key regulator of the intrinsic death pathway that amplifies caspase-dependent and caspase-independent execution of neuronal apoptosis. Therefore pharmacological inhibition of BID provides a promising therapeutic strategy in neurological diseases where programmed cell death is prominent. BID is activated by Caspase 8 in response to Fas/TNF-R1 death receptor activation. Activated BID is translocated to mitochondria and induces cytochrome c release, which in turn activates downstream caspases. BID action has been proposed to involve the mitochondrial re-location of its truncated form, tBid, to facilitate the release of apoptogenic proteins like cytochrome c.

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